In the Claims

Claims 1-17 (Cancelled)

Claim 18 (Original): A method for testing a compound suspected of promoting or inhibiting phosphorylation of one or more proteins related to Alzheimer's disease, said method comprising: providing a mammalian cell; administering to said cell antichymotrypsin and said compound; and monitoring the phosphorylation state of said one or more proteins.

Claim 19 (Previously amended): The method of claim 18, wherein said protein is tau, APP, cdc-2/cyclin B, cdk5, p53, cdc47, MAD, cyclin D, or cyclin E.

Claims 20-21 (Cancelled)

Claim 22 (Previously amended): The method of claim 18, wherein said cell is neuronal.

Claims 23-42 (Cancelled)

Claim 43 (Currently amended): A transgenic mouse whose genome comprises at least one transgene comprising a nucleic acid sequence encoding ~~a protease inhibitor~~alpha-1-antichymotrypsin (ACT) operably linked to a glial fibrillary acidic protein (GFAP) promoter effective for expression of said nucleic acid sequence in the brain tissue of said transgenic mouse, wherein ~~said protease inhibitor interacts with amyloid beta peptides within the brain tissue of said transgenic mouse, and wherein said protease inhibitor is selected from the group consisting of antichymotrypsin (ACT), antitrypsin, and alpha-2 macroglobulin.~~ when said transgenic mouse is crossed with a second transgenic mouse whose genome comprises a nucleic acid sequence encoding an amyloid precursor protein (APP) V717 mutant or whose genome is homozygous for a non-functional apolipoprotein

(ApoE) gene, progeny are produced having an increased rate or extent of amyloid formation within the brain tissue.

Claims 44-46 (Cancelled)

Claim 47 (New): The transgenic mouse of claim 43, wherein said GFAP promoter is devoid of ATG start codons.

Claim 48 (New): The transgenic mouse of claim 47, wherein said nucleic acid sequence encoding ACT is expressed in astrocytes within the brain tissue of said transgenic mouse.

Claim 49 (New): The transgenic mouse of claim 43, wherein the genome of the second transgenic mouse comprises a nucleic acid sequence encoding the amyloid precursor protein (APP) V717 mutant, and wherein the progeny have an increased rate or extent of amyloid formation within the brain tissue.

Claim 50 (New): The transgenic mouse of claim 49, wherein the nucleic acid sequence encoding the APP V717 mutant is operably linked to a platelet-derived growth factor (PDGF) promoter.

Claim 51 (New): The transgenic mouse of claim 43, wherein the genome of the second transgenic mouse is homozygous for a non-functional apolipoprotein E (ApoE) gene, and wherein the progeny have an increased rate or extent of amyloid formation within the brain tissue.

Claim 52 (New): The transgenic mouse of claim 49, wherein said genome of said second transgenic mouse is homozygous for a non-functional apolipoprotein E (ApoE) gene, and wherein said progeny have an increased rate or extent of amyloid formation within the brain tissue.

Claim 53 (New): A transgenic mouse whose genome comprises at least one transgene comprising a nucleic acid sequence encoding alpha-1-antichymotrypsin (ACT) operably linked to a glial fibrillary acid protein (GFAP) promoter effective for expression of said nucleic acid sequence in the brain tissue of said transgenic mouse, wherein said genome:

(a) further comprises a second transgene comprising a nucleic acid sequence encoding an amyloid precursor protein (APP) V717 mutant; or

(b) is homozygous for a non-functional apolipoprotein (ApoE) gene; or

(c) further comprises a second transgene comprising a nucleic acid sequence encoding an amyloid precursor protein (APP) V717 mutant, and is homozygous for a non-functional apolipoprotein gene;

and wherein said transgenic mouse has an increased rate or extent of amyloid formation within the brain tissue.